# GOUT

*Dr. WADE*

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## AS A PERIPHERAL NEUROSIS



BY

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## CHAPTER I.

IT is desirable to state the scope and object of this essay. To do this clearly and briefly it is necessary to glance at the theories which have been or are current in regard to gout, of these there are broadly three. Firstly that which refers it to impurity of the blood, or, to maintain an old word which corresponds so little to modern conceptions as to have become almost obsolete, the humoral theory. Secondly the neural or nerve theory, which views gout as depending upon a disordered condition of the nervous system. Thirdly the neuro-humeral theory, which combines the two first and regards gout as being due partly to disorder of the blood and partly to disorder of the nervous system.

Much as we know about gout, it would be idle to deny that there are still wide gaps or

*lacunæ* in our knowledge, many questions which we cannot at present answer. It is impossible to foresee what answer science will give, when she is able to give any, to these questions. It is therefore impossible to foresee in what way or to what extent these answers, when they come, will necessitate a new theory, or a modification of any one that we at present form.

From the standpoint of our present knowledge it seems to me impossible to be satisfied with any theory less comprehensive than the neuro-humeral one.

It may here be justly remarked that "the nervous system" is too vague a term, for this system consists of collections of grey matter in the brain, in the medulla, in the cord and in numerous ganglia; of white matter which is in immediate apposition with the various grey foci, and of white matter which extends to the more distant parts of the body as nerve trunks, and of the minute endings of these in the tissues. "Surely," an objector may say "you ought if you ask me to accept your nerve-theory to tell

me in which one of these various parts you locate the disease and for what reasons you fix on that particular part." The nearest approach to an answer which has been given to this fair objection is that it is "some nerve centre." Now I am not prepared to deny that this may be so. On the contrary, it would seem to me to be flying in the face of some of the most certainly ascertained clinical facts to deny that the highest centres may be concerned in the production of gout in particular instances. Because few facts in connection with gout are more certain than that excessive brain work, and care and anxiety are in some instances the apparent producers of the gouty attack, though whether by augmenting the gouty poison in the blood or by some direct action on the part which is attacked is by no means so certain. There are truly no lower centre or centres of which, with certainty, as much can be said. But it would be fatuous to contend that evidence exists to show that the higher centres are in all cases the seat of origin of gout whether directly or indirectly.

At all events I certainly do not do so. So that we are confronted with two facts. One that there are a multiplicity of phenomena which indicate that the nervous system is implicated in gout. The other, that we seek in vain for any precise indication that any one portion of the central nervous system can be identified as being, whether directly or indirectly, that which is so implicated, except in an occasional way. It was the pressure of this dilemma that caused me to turn my attention in another direction.

A frequently occurring, and a most obvious feature of gout is its invasion of the joints, and the great numerical preponderance of instances in which the great toe is attacked as compared with any other joint. Any theory of gout which does not explain this superior frequency must be stamped with inadequacy. On the other hand any one which does, at once establishes a claim to attentive consideration.

An investigation of the anatomy of the joint only served to corroborate Ebstein's *dictum*,

that there is no difference between it and any other joint in the body. That is to say no minute nor any coarse structural difference which throws light upon its pathological reaction to the poison of gout. But on the other hand I was struck by certain features of its environment, which we have all known since we were students, but to which we have never given heed. Looked at from the point of view in which I then stood they assumed a possible importance. They recalled to my mind an isolated clinical observation of previous years and they determined me to institute a more searching and minute examination of living cases of the disease. It is the result of such investigations and the inferences to be drawn from them that are related in subsequent pages.

## CHAPTER II.

WE are familiar with the swelling, redness and extreme sensitiveness of the skin over the toe-joint in acute gout. We know also that a similar condition is observed often to exist in the neighbouring skin and soft parts.

As this condition subsides a time comes when the patient will, though reluctantly, allow the part to be cautiously handled. It is a good many years since it became known to me, that the tenderness did not like the redness and swelling abate equably over all the surface. In some places the skin remained tender in others not. The tenderness had a "patchy" character. There was not always a corresponding difference of appearance in the skin. This long remained to me an isolated clinical fact, unexplained though of pretty constant occurrence.

Subsequently, as I have said, my attention was again attracted to this subject and I investigated it more closely. I then found that the residual tenderness might not only be in the shape of spots and patches, but also in that of lines. I will cite as examples of this latter condition two cases which have been under my care this year.

One was a lady, aged about sixty, for twelve years subject to attacks of acute gout in the great toes and other parts. Her heart, lungs and stomach were failing and she died a few days after my only visit. I found that within a few days she had had gout in the left toe which had entirely recovered. Before it was well gout had come on in the right toe, which was still swollen and painful. From the base of the toe for about one and a half inches there extended a narrow line of acute tenderness. There was no super-jacent redness or swelling. Pressure on each side of this line was *absolutely* painless. The line itself was apparently equal in breadth to the diameter of an ordinary sized steel knitting pin.

The second case occurred before the one above described.—

A gentleman, aged fifty-eight, had a first attack of acute gout in the left great toe, extending also a little above it. After a few days' treatment this abated, and the great toe-joint admitted of passive movement in all directions, the opposing surfaces of the two bones being at the same time firmly pressed against each other. But a line of tenderness, similar to that described in the former case, remained. It started from the base of the great toe and extended slantingly across the foot as far as the centre of the bend of the ankle. The tenderness disappeared at the rate of about three-quarters of an inch a day from above downwards. There was during this period no diminution of the tenderness in the part which remained tender. For the sake of clearness I have described this case as it appeared after all redness and swelling had disappeared. For the sake of accuracy I must now state that the line of tenderness existed while the redness and

swelling still remained over its distal part. The upper portion lay in a part which had not been red, swelled, or painful.

We have now to determine the anatomical seat of this tenderness. If we refer to Swan's "Demonstration of the nerves of the human body," we find (plate xxiv, fig. 2,) a nerve which he describes thus "No. 4 Inner division of the dorsal branch of the peroneal nerve, giving filaments to the foot, the inner side of the great toe, the outer side of the second and the inner side of the third toes." But if we look again at Swan's plate we find that the nerve No. 4 divides just at the middle of the lower border of the anterior annular ligament into two branches. Of these one runs slantingly across the foot to the base of the great toe and it is this branch which is distributed to the great toe. The other branch is distributed to the other toes.

According to this authority then, this nerve follows a course which coincided with the line of tenderness, the breadth of which, so far as we

can judge, corresponded with that of the nerve. The tendon of the long extensor of the great toe runs a somewhat similar course but its width is several times that of the nerve. So much so that I cannot think that, if it were tender, the tenderness could with any accuracy be spoken of as a "narrow line." Voluntary movements of the great toe in the second case were quite painless, which is probably inconsistent with an acutely tender state of the extensor tendon.

The veins over an acutely gouty joint no doubt often appear to be tender. Whether this tenderness is intrinsic or merely results from the distension affording greater resistance behind the skin when this is struck, and so enhancing the skin tenderness it is not needful here to enquire. For there was no venous distension present in either case at the time spoken of.

I submit then that there is good ground for the conclusion that the nerve was the anatomical seat of this tenderness.

Two further questions of great importance now arise. Firstly what is the nature of this neuropathy? Secondly what is its relation to the gouty joint? The consideration of these questions will be conveniently postponed till after attention has been called to some other clinical facts.

Gout as affecting the hands, may as regards its acute, subacute, or chronic phases, be divided into five classes. First—Gout in the knuckles: Second—In the back of the hands: Third—In the ball of the thumb: Fourth—In the hypothenar region: Fifth—Those cases in which two or more of the previous classes occur at the same time.

My first observations in this clinical division were made upon myself, for I have twice had gout in the root of each thumb, and once in the right hypothenar region. But only two of these attacks have occurred since my attention has been directed to these investigations, and I was at the same time sufficiently well to investigate my symptoms. In the hypothenar attack the

pain and tenderness in the part itself were, though quite distinct, by no means severe. But on examination I found that there was extreme tenderness on pressing the wrist, just to the radial side of the pisiform bone. The pressure left an aching at the part where it was applied, and produced for some hours an increase of the pain and tenderness in the hypothenar region. Pressure here, in healthy persons, produces a contraction of the abductor muscle of the little finger. This is not absent when in gouty persons there is also tenderness on pressure. From this, as well as from anatomical considerations, it is to be inferred that it is the ulnar nerve which is the seat of this tenderness. Here is another instance: Meeting in the street a gentleman, æt. fifty-two, who has for some years suffered a good deal from acute joint gout, he told me that during the preceding night gout "had come out" in his hand, indicating the hypothenar region. I said give me your hand. As I was slipping my thumb under his cuff he said "Oh no its not there, there is nothing

there." By this time I had my thumb in position and gave a squeeze. He snatched his hand away, exclaiming in a voice too loud for the street "Oh, by gad there is though!" and added "I never felt anything there till you pressed it." I have mentioned this case in somewhat trivial detail to show that there was no collusion or self-deception.

A medical man æt. seventy-two had for several weeks remittent and chiefly nocturnal pain and swelling in the knuckles, and painfulness on usage, of the right hand. He was much surprised on finding that pressure at the point indicated was extremely painful and that the pain remained there for some hours after. He was also an example of the next form of tenderness, namely, that in connection with the thumb. My first observation of this form was made on myself. There was a good deal of pain all round the ball and root of the thumb, with redness and some swelling, movement was painful. Keeping the bones straight and holding the first phalanx no pain was produced

by pressing firmly and moving the joints, so far as that could be done withuot disturbing the soft parts. Pressure on the wrist in the interval between the tendons of the two extensors of the thumb was acutely painful, it also much aggravated the painfulness of the thumb for some hours after. It is to be noted with regard to the case of the medical man at seventy-two that just at the wrist-joint the ulnar nerve gives off a deep branch which supplies *inter alia* the joint between the metacarpal bone and the first phalanx of the ring finger, of the second finger and of the fore-finger (Swan, pl. xxii, fig. 2, Nos. 28, &c.) I have also found tenderness in the nerves which run one on each side near to the dorsum of the fingers and one on each side of their palmar aspects. If there should, as there may, be general tenderness of the fingers, the sites of these nerves cannot be distinguished. So far as I have been able to test it, and that has been in about a dozen consecutive cases, tenderness will almost certainly be found in one or more of the situations indicated when there

is or recently has been gout in or flying about the hand or fingers. There is also another place where tenderness may be found. This is about the centre of the dorsum of the wrist, just where Swan shows a branch of the ulnar nerve, joining one from the radial.

Let us now turn to another division of the subject. Persons who are in a gouty condition are subject to pain and tenderness, recurring at irregular intervals and of very variable, sometimes persistent, sometimes of only momentary duration, about the ball of the great toe. This tenderness is specially felt in walking. This pain is, by common consent and repute, located in the metatarso-phalangeal joint of that toe.

This view receives *primâ facie* support from physical examination. For undoubtedly pressure applied over the upper aspect of the joint, or at its side, or on the lower aspect of the joint may elicit a complaint of tenderness in one or more of these situations. I myself have for years been from time to time subject to this affection. And for years in dutiful submission to current

teaching looked upon the joints as the seat of the trouble. The circumstances previously stated led me to investigate the matter impartially and *de novo*. What did I find? In the first place that passive movement of the joint surfaces upon themselves, the first phalanx held firmly between finger and thumb and all possible pressure made upon the joint was absolutely painless. There was no evidence of fluid in the joint. It is impossible without cutting into it to obtain stronger evidence that the joint was healthy.

On critical examination the tenderness was and will be found as above mentioned in one or more of three situations. If we look at Swan's demonstrations (pl. xxiv., fig. 2), we find that two branches of the internal plantar nerve lie one to the outer side and one to the inner side of the plantar aspect of the great toe. I have demonstrated to myself on myself many scores of times that what appears on off-hand examination to be tenderness in the great toe-joint is really one or two lines of tenderness occupying

the site of these two nerves. When there is only one line it is commonly, but not invariably, the inner one Between these two lines is to be felt the bone absolutely indolent. No doubt if we press over the joint we shall find tenderness, because the nerves run across the joint. But we shall also commonly find an equal degree of tenderness in both the distal and the proximal sections of these nerves quite away from the joint. With regard to the tenderness over the superior aspect of the joint enough has been said in speaking of it in acute gout. Only adding that it occurs in the absence of that disorder. The side tenderness I have found not only under the circumstance with which we are now dealing but also on the abatement of acute gout. It is I think probably situated in branches of the peroneal (so called by Swan and by Quain and Ellis external-popliteal) nerve. Or it may be in the saphenous nerve. Probably the former, as it has not appeared to me to have sufficient longitudinal extension to be situated in the latter.

There is another part in which I have found tenderness. In most accounts of gout we find mention of pain and tenderness in the sole of the foot as occurring in certain cases. This tenderness is always described as being situated in the plantar fascia. Nor would I deny that such may be the case. But on the other hand I have found a line of tenderness near the inner margin of the sole of the foot. On referring to Swan (pl. xxv., fig. 3, No. 8), we find that just in this situation lies a slender nerve which he thus describes. "A branch of the branch five of the inner plantar nerve, to terminate in the joint of the great toe between the metatarsal bone and the first phalanx." "Branch five" is the nerve which has been referred to as running on the inner side of the inferior aspect of the great toe. It may be argued that if the inflammation of the great toe-joint depended upon a morbid condition of this nerve, it should in all cases be found to be tender. I shall deal with this argument hereafter.

I have repeatedly verified in other persons the fact that tenderness supposed to be in the

toe-joint was really located in one of these nerves and not in the joint.

Pains and tenderness in the thick of the heel—I am not now alluding to the tendo Achillis—are common in gouty persons. Few old gouty patients are not familiar with their occurrence from time to time, though they often do not think enough of it to mention it to the doctor. The outer half of the heel receives its nervous supply from a nerve compounded of a "communicating tibial branch of the sciatic nerve" and a "cutaneous branch of the peroneal nerve, receiving the communicating tibial" (Swan, pl. xxv., fig. 2, Nos. 20 and 21). This joint nerve in its course lies behind the outer ankle.

The inner half of the heel is supplied by the posterior tibial which lies behind the inner ankle.

Now I have often noticed in myself the following facts, which however I have not yet had an opportunity of verifying in other cases. The pain commonly of an aching, sometimes of a burning, character, a superficial and also a

deep-seated tenderness, may occupy either one lateral half of the heel or both halves at the same time. In the latter case they may not be of the same intensity on each side. Distinct tenderness may be correspondingly present behind one or both ankles, presumably in the nerve-trunks above mentioned. But such tenderness is not invariably present.

Tenderness in all the above mentioned situations, both in the feet and in the hands and wrists may from time to time be found even when the patient has not felt it during their ordinary use. Its presence is of importance as, in my opinion, identifying as gouty, visceral or other symptons which we should otherwise only with uncertainty suspect to be so.

These pains are notoriously fugitive, often a mere momentary dart. The tenderness also is often inconstant, present and absent in the course perhaps of half an hour; or if not absolutely vanishing yet varying much in intensity. This I have repeatedly observed in myself.

I desire now to turn to another matter, namely the connection of articular gout with injury. This connection is well known, but I am not sure that it habitually receives as minute attention as it deserves. I shall briefly catalogue the various circumstances under which it presents itself.

We may divide these cases into three classes. First—Gross mechanical injuries such as sprains, bruises or blows. Second—Minor mechanical injuries. Third—What may be described as physiological injuries. Of the first class we have several varieties. First—When a first attack of gout appears in a part long after the injury has occurred. A clergyman sprained his right ankle badly in running down stairs. It was not till fifteen years after that he, being hereditarily gouty, had an attack of gout (traceable to worry) and that was in this ankle. Second—cases in which gout immediately or very shortly follows the injury and is confined to the injured part. This happened to a gouty surgeon who not very severely sprained his right knee

by being thrown from his tricycle. In two or three days the knee, in which there had remained a little pain from the time of the accident became the seat of an acute and very long continued attack of gout. Third—Cases in which gout appears in other joints as well as in the injured one. A gentleman æt. fifty who had had, during the previous twelve years, several severe attacks of gout, twisted his ankle, it remained very painful and soon became swollen, during the night the other ankle also became swollen and painful, and he then recognised that he had gout in both joints. This opinion was confirmed by the Italian doctor whom he called in, he being at the time at Turin. He was confined to bed for six weeks. Another gouty man of about the same age fell and twisted his knee, it very shortly after was seized with acute gout. This migrated to the other knee, subsequently to each ankle, and finally returned to the knee first affected. He had a long and troublesome attack, lasting many weeks. A lady æt. eighty-four, who was of a

gouty stock, but had never had gout, was thrown from a high dog-cart. She did not know exactly how she fell, but not on her back. In two or three days she had very acute gout in both wrists and subsequently in both elbows.

Fourth—Cases in which there is a long interval between the accident and the gout, but not entire freedom from symptoms due to the accident. While getting into my carriage with the right foot on the step the horses gave a start. From that time with variations in degree, but rarely if ever complete absence, my knee was more or less but never severely painful on movement. The pain was in a transverse line just above the joint and there was also tenderness. These symptoms lasted for ten months. At the end of that time I went to Buxton and put myself under the care of my friend Dr. Robertson. The baths relieved without entirely curing the knee and quite relieved the general arterial tension which had been all the time unduly high. Within a week of my return home acute inflammation of the

knee-joint, with great effusion, occurred. It is worth mentioning that at Buxton in a bath at 98° the water appeared to the knee to be several degrees colder than it did to any other part of the body. When I have pain in the heels hot baths, whether whole or partial, generally feel hotter to the heel than to other parts. This form of altered sensibility, which I have known to occur in other persons points to an abnormal state of the sensory nerves.

Fifth—I am not cognisant of any case in which injury to one joint has appeared to cause gout not in it but in another.

The second class comprises those cases in which minor injuries appear to cause gout. It is hardly necessary to point out that the distinction between "gross" and "minor" injuries is of a somewhat arbitrary character. These cases present many varieties.

First—Production of gout by the use of parts, as of the thumb from writing, or from repeated use of it for lifting purposes. Of both these I have had experience in my own person.

Second—Aggravation of gout by use of the part. A widow lady was much addicted to crochet work. She used to have attacks of gout in the right hand, which unless or until they were so severe as to make it impossible she did not allow to interrupt her work. For two years her left arm and leg have been absolutely paralysed. During that time she has often been in a gouty condition and has several times had gout in the right hand, but never at all approaching in severity those she used to have previous to the paralytic attack.

Three—Reproduction of gout by use of the part. A gentleman fond of and accustomed to pedestrian exercise has had many attacks of acute gout in various parts. On two occasions when the feet and ankles had been affected, he, when he thought himself sufficiently recovered, walked several miles in heavy boots. On both occasions acute gout was reproduced in the parts which had been affected.

Four—Production of gout by pressure. The familiar examples of this are found in cases

where gout in the great-toe is produced, aggravated or reproduced by the pressure of a too tight boot. Here is an example of a different kind, which occurred in my own person. At a certain stage of my knee attack all symptoms had disappeared except the swelling, which was great and prevented the knee from being flexed. I had occasion (before I had left my bed) to drive for about an hour into the country, see a patient and drive back again. In my carriage, in order to avoid any discomfort to the joint, I stretched the sound leg straight out and placed the other limb upon it as upon a splint. The outside of the right heel rested upon the dorsum of the left foot towards its outer edge. I came back and went to bed, none the worse as regards the knee. But in the course of an hour I felt pain in the outer side of the right heel. On examination it was found to be tender and brightly red over a circular space larger than a half-crown. This condition took three days to disappear. This it may be said is a trivial instance of gouty

inflammation. That is so. But any adequate theory of disease must be able to explain trivial just as much as the most portentous symptoms. It may be asked, why, action and reaction being equal and opposite, no effect was produced on the dorsum of the left foot? Without undertaking to answer that question in full it may be pointed out, First—That the tissues of the heel are dense and tense, while those of the other part are lax. Second—That gout is common in the heel, very rare in the other part. Third—That gout was already manifested in the right limb and not in the left.

It is generally recognised that gout may be produced in joints by cold, and by wet feet. And if producible it may be aggravated or reproduced by the same agency.

Five—If such disorders as bronchial gastric or intestinal catarrh may be gouty, as many physicians besides myself think, there can be no need to plead that cold may be an element in their production as it is in non-gouty cases.

Six—Neuralgia and neuritis as distinct and substantive disorders which are not always

distinguishable the one from the other are by recognised authorities with whom I am in entire accord, attributed in certain instances to the gouty condition, or, to use another phrase, to the gouty poison. Cold is often a factor in their production.

Seven—In coming to such disorders as gout in the stomach we are upon more debatable ground. Sir Thomas Watson said he had often found that "gout in the stomach" resolved itself under the influence of an emetic "into pork" in the stomach. To those who believe that gout may be produced in the toe by the pressure of a tight boot this *dictum* would go to prove, not that these attacks were not gout, but that they were gout, and gout produced by a sufficient and ascertainable cause, viz., a local irritant. That certainly is the effect produced upon my mind.

Before leaving this part of my subject one or two remarks. Is it not the case that external agencies have not been sufficiently regarded as gout producers? That these cases have been

looked upon rather as exceptions and oddities, without any special or deep significance? If so, that, insufficient inquiry has been made into the minute history of gouty cases in respect to previous or coincident injuries or other external influences? Finally, if so, might not a more constant and minute inquiry reveal a much more constant relation between such external circumstances and the appearance of gout in a particular part?

## CHAPTER III.

WE now have to enquire into the nature of the morbid condition of the local nerves which is so frequently to be found in the vicinity of gouty manifestations, whether these be acute and severe or subacute and slight.

Let us consider to what known pathological conditions this affection is most akin, having regard to its clinical features. It is obvious these resemble those of neuralgia, hyperæmia of the nerves, and neuritis both acute and chronic. Neuralgia has no pathological anatomy, for the view which has been supported by some authorities, viz —that it is in all cases due to neuritis is not generally accepted. At the same time it has an undoubted clinical existence, and in some respects the features of this gouty neuropathy resemble those of neuralgia. But

little is known about hyperæmia of the nerves or its actual clinical occurrence. Erb remarks on hyperæmia of the nerves, that "it appears to be deserving of some attention in the future, since, perhaps, many so called functional neuroses are referable to it."

Of neuritis both acute and chronic much is known both clinically and pathologically. Both neuralgia and neuritis are recognised by the best authorities as occurring in gouty persons and as being actually due to gout. Nor are they as substantive diseases, that is as existing independently of joint-gout at all rare affections. An attempt therefore to give a new significance to neuritis and to extend its domain into parts where its existence has not hitherto been recognised, is in accordance with, and not hostile to, facts already known and accepted.

It will not be useless to state now some of the facts of the natural history of acute neuritis. And these I shall take almost verbatim from the excellent article by Professor Erb in Von Ziemmsen's Cyclopœdia of the Practice of Medicine.

Etiology. The most frequent and best known causes of neuritis are wounds of various kinds, contusion, rupture, &c. Slight mechanical injuries may also cause neuritis, such as a blow on a nerve trunk, strong compression of a nerve, severe concussions of the nerves from long travelling in an ill-constructed wagon, sudden and violent muscular movements, violent efforts to raise heavy weights, &c. It also arises from "catching cold," exposure to a draught of air, &c. Inflammations of various organs constitute a very common cause of neuritis. After acute diseases neuritic processes are not unfrequently developed. Lastly, no one can wonder that it is impossible in many cases to discover the real cause of neuritis, and hence that we must speak of its origin as spontaneous. It appears moreover, as if many persons have a special predisposition to neuritis, and especially as if the disposition for the disease to spread upwards or downwards along the nerves is present in very different degrees in different individuals. Special attention must be paid to this point in

future." As regards the pathology of neuritis much does not require to be said. In the earlier stages there are microscopical evidences of inflammation in the neurilemma, then incipient disintegration of the medulla and finally of the axis cylinder. It may be taken that it is not in all cases of neuritis that the inflammatory process reaches to this last stage, and restoration of the nerve may be established. We find that there are two classes of change in these cases. The one affecting the protective coverings of the nerve; the other affecting its conducting apparatus. The two classes of change give rise to two classes of symptoms. The one resembling those common to inflammatory affections, the other due to interference with, or abolition of, the functions of the nerve as a conductor of force. These vary according to the special endowments of the nerve affected. When they are of a trophic or vasomotor character, the changes are also of that character, It remains to mention one other feature of neuritis. In whatever part in the course of a nerve it

originates, it has a tendency to spread both downwards towards the periphery *(neuritis descendens)*, and upwards towards the centre *(neuritis ascendens)*. But according to all authorities the ascending is much more common than the descending form of extension. It is also believed that without any upward extension of the actual inflammation an *influence* may ascend from the inflamed part upwards to the cord, disturbing its functions and even causing myelitis and its ordinary results. I will not go into details respecting the chronic form of neuritis, which may either be a primary affection, or secondary to an acute attack. But it may be well to draw the readers attention to certain of the clinical features of neuritis and neuralgia which find a distinct parallel in those of gout.

Origin from injuries, and from cold.

Intensity of the pain.

Intensity of the tenderness over both the trunk of the nerve and its peripheral distribution.

Pain and tenderness, the earliest symptoms.

Radiation of pain towards periphery.

Remissions of pain.

Exacerbations tend to be nocturnal.

Pain augmented by every movement of the part.

Cramps of muscles.

The (as a rule) more intense and early involvement of sensory, as compared with motor, nerves.

Sensitiveness to pressure constant in neuritis. Not constant in neuralgia.

Trophic disturbances of skin, nails and joints.

Hence glossiness of the skin in cases of long standing.

Vasomotor disturbances.

It must not be forgotten that the conductivity of the nerves may be impaired in quite another way than by inflammation of the neurilemma.

Certain poisons introduced into the blood are capable of producing a degeneration of the white substance of Schwann and of the axis cylinder. Of this the Diphtheria poison is an example. It would be more correct to say the Diphtheria poisons, for there are two, an albumose and an

organic acid. Each of these, the albumose more actively than the organic acid, is capable of disintegrating motor, sensory, and sympathetic nerves. Their action is firstly on the white substance of Schwann and secondarily on the axis cylinder, the neurilemma where that exists is but slightly if at all affected. Such at all events are the conclusions drawn by Dr. Sidney Martin, from his admirable investigations as detailed in his Goulstonian Lectures (1892).

It seems to me probable that the painlessness of Diphtherial paralysis is due to the absence of changes in the neurilemma

It is not necessary to enumerate the many features of gout which support the idea, originated by Cullen, and now generally entertained, that there is an intimate connection between this disease and the nervous system.

This is not a treatise on gout but an effort to justify the view that, granting this connection, it is in the peripheral nerves we must look for the real and chief bond of union. I venture to think that what has been said will lead the

reader to feel that after all it would not be very surprising if this view should turn out to be correct.

## CHAPTER IV.

TO give reasons, acceptable as it is hoped they may be, for thinking that there is no inherent improbability, but the reverse, in the neural element of gout being principally a disorder of local nerves is not sufficient, it is necessary to give reasons for thinking that it is.

Let us examine the two first cases which I have described. One was a case of first attack, in the other there was a history of many previous attacks, of genuine gout occupying its ordinary seat, the tarso-metatarsal joint of the great toe. In each there was evidence which would under any other circumstances be accepted as proof of the existence of neuritis in close proximity to, indeed at one extremity terminating at, the joint. Why

should this evidence not be accepted as equally conclusive in these cases? It is a fact, at all events it is accepted as one by all the best modern authorities that neuritis occurs as an isolated and distinct affection in gouty persons. It is believed by the same authorities that it is a manifestation of the gouty condition or poison. In short, that there is truly such a thing as gouty neuritis Is there then any inherent improbability that it may occur not only as an isolated affection but also in close connection with another local gouty inflammation, that namely of a joint? Quite the contrary is the case. "Inflammations of neighbouring organs, extending to nerves traversing or adjoining such organs, constitute a very common cause of neuritis." (Erb). The fact then that neuritis is to be found not only as an isolated affection, but in close connection with the ordinary acute joint-affection of gout, is important. The bringing into close relation and proximity these two reputedly separate affections is a distinct step in an argument aiming to show that one is

dependant upon the other. But it is quite inconclusive as to which is the primary and which the secondary affection. Indeed if it points to any conclusion, it is to one which is quite in consonance with current opinion and accepted facts, viz.: that the neuritis is a secondary inflammation set up by a primary inflammation of the joint.

But now let us turn to the consideration of the other category of cases. It will probably not be disputed that in the common opinion they do not differ from acute attacks in kind but only in degree. But what does an examination more searching and minute than ordinary reveal? The presence of neuritis, the absence of arthritis. The presence of a nerve disorder, the absence of a joint disorder. The prior presence of a nerve disorder of a kind which is admittedly capable of producing sensory, vaso-motor, and trophic changes in parts which as a nerve it supplies. The subsequent appearance of such sensory vaso-motor and trophic changes. There is here—it is right to point out—a link

at present missing. We may easily find instances in which an acute arthritis has been preceded by the minor pains and tenderness which are, not in the joint as has hitherto been assumed, but in adjacent nerves. And I have seen cases (two recently), in which there was at the same time gouty affection of the thumb without arthritis and gouty affection of the fingers with arthritis and effusion into some of the knuckle-joints.

We want to trace a pre-existing neuritis actually culminating in a frank attack of arthritic gout. We can hardly expect to have an opportunity of seeing this in our ordinary patients. But there are many gouty medical men. Is it too much to hope that they will summon up courage enough to examine themselves, or to permit some one else to examine them with minuteness in the first onset of acute gout. The point to be determined is this. Is there tenderness in the skin or in some of the nerve-trunks surrounding the toe-joint at a time when there is no tenderness in the toe-

joint itself? The thumb or indeed any other joint may be put to the question in a similar manner.

It must however be remembered that such an examination may be inconclusive. A neuritis need not extend far from the joint, for the effects of the suspension of nerve conduction are the same in whatever portion of the individual nerve fibre the obstacle occurs. Or indeed if as in the arthropathy of tabes the nerve influence is affected at its source. And if the skin to which it is distributed be at the same time very tender it may be impossible to determine whether the pain is in the skin or in the joint. Still more is this the case when the minute trunk of a nerve itself is inflamed and lies near the joint, for a slight movement of the part may be, indeed often is, extremely painful though the joint be evidently not affected. But in other similar cases it may be impossible to say for certain that the pain is confined to the soft parts and not also partly in the joint.

I hope indeed that all the clinical statements upon which my argument is based may be rigorously tested by those who have the opportunity.

I venture now to claim to have established a high degree of probability that in gouty arthritis the joint affection is secondary to and caused by a preceding (though possibly only by a very short time) affection of the peripheral nerves From this point of view a neuro-humoral theory of Gout should be stated thus :—

In certain individuals, many of whom have a parentage members of which have been similarly affected, aberration of proteid metabolism occurs. Its causes, origin and stages are unknown. The most manifest of its terminal products is uric acid. This in the form of quadriurate of soda is present in the blood in abnormal quantity and lowers its alkalinity. It is suspected, though not decisively determined, that there may be also present other terminal, collateral, or bye products.

The circumstances under which this hypo—or para—metabolism happens are excessive mental

strain especially if consisting of or accompanied by anxiety or worry, excessive use of certain kinds of food, or of alcohol, especially of fermented malt liquors, or of strong or highly saccharated or hyperacid, or effervescing wines or those with a high percentage of ætherial products. In consequence of this blood-state, the stability of the nerve-trunks is impaired; and therefore their power of resisting external influences which would be inoperative upon normal nerves. These external influences are the ordinary and still more the extraordinary, use and effort of parts, concussion, and compression of the nerves themselves, injury or strain, irritants or cold.

The combined result of the intrinsic state of the nerve trunks and of extrinsic influences is the production of further changes in the nerves notified by symptoms which are those either of neuralgia or of neuritis, or of both, but the anatomical basis of these has not as yet been investigated.

The further effects of this neuropathy depend first, on its intensity, second, on the endowments

of the nerve affected, viz., sensory, vaso-motor, trophic, or motor, third, on the territorial distribution of its ultimate branches, fourth, on the involvement of two or more of the above four classes of nerves.

The blood poison seems to have an elective affinity for the various classes of nerves in the order in which they are above stated.

An increase, especially if it be sudden, in the blood dyscrasia also enhances the neuropathy, and may possibly be able to induce it without the co-operation of any external cause. Such increase may be due to—

First—Aggravation of the primary cause of the altered metabolism and consequent increase of morbid products.

Second—Liberation into the blood of such products which had been previously stored up in some part or viscus.

Third—Diminution of the eliminative power of some organ or organs by which these products had been previously separated from the blood.

It is probably under one of these heads that is to be classed a general perturbation of the system.

The neuropathy has the following qualities in common with neuritis, that of ascending or descending from its primary seat, unless that be in the ultimate fibrils when it necessarily has an ascending power only. In common with neuritis and neuralgia, that of affecting the state of the spinal (and possibly of higher,) centres and radiation thence to other nerve territories.

The highest and lower cerebral centres must be credited with the power of originating or aggravating the general gouty condition. Conceivably through their known potentiality over recognised seats of metabolic activity. They may perhaps also be credited with the power of determining local outbreaks. Conceivably through their known potentiality over peripheral especially vaso-motor nerves. The altered effect being due to the altered conductivity of the affected peripheral nerve.

The high cerebral centres are also liable to be affected by the blood dyscrasia (e.g., ill temper, melancholia), but in what way this is brought about there is no evidence to show.

There is no doubt that a biurate salt may be deposited in some of the viscera affected by localised gout. Such a deposition is usual when the part affected is a joint. This is by no means a necessary or invariable result, according to the only evidence at our disposal. For while on the one hand the deposit has been found many years after a joint had been affected once only, on the other none has been discoverable in a joint which had been many times affected. This deposit must be looked upon as an epiphenomenon. And its presence is probably due to the peculiar mode in which joints are nourished. The amount of deposit is, speaking generally, in inverse proportion to the acuteness of the other symptoms, and therefore less in the earlier attacks and greater in the later ones. It may also take place in the absence of any preceding or concomitant pain, or redness, or of any swelling except that which itself produces. An attack in a joint is sometimes followed by a cessation of extraneous symptoms which we suppose to depend upon the abnormal state of

the blood. When this is so it would appear that it is either evidence, or the cause, of a reversion to normal metabolism, but in what way this result is produced we have no evidence.

## CHAPTER V.

A THEORY however specious is valueless except in as far as it is based on facts, which it not only recognises but harmonises. It may I think be fairly claimed for this theory that it harmonises and brings into line a greater number of the admitted facts of Gout than any other. It does not fail to recognise that there are important elements of the disease the nature of which is as yet concealed from our eyes. But it is not in hostility to any admitted facts.

It recognises two great classes of facts.

First—Those which have for ages been seen to indicate that the fluids of the body are concerned in the production of gout and which are therefore the basis of the humoral theory.

Second—Those which indicate that the nervous system is concerned in the production of gout, and are the basis of the neural or nerve theory of gout.

This latter since the time of Cullen has always held a distinct though fluctuating hold of medical opinion. Of late years these two at one time opposing theories have been more or less held in conjunction. As indeed it seems to me they must be, unless we ignore some of the most prominent features, and best ascertained facts of the disorder. The weakness of the nerve theory has hitherto been its vagueness. And this has materially retarded its general acceptance. To this hitherto almost abstract proposition the localisation of the nerve element in a definite section of the nervous system gives concreteness. To many minds "the nervous system" in this relation, means nothing. Nothing that is which they can grasp. There is at all events no such difficulty in regard to neuritis, as we may at least provisionally term it. For the first time a tangible basis is given

to the nerve theory and thus should commend my theory to the holders of that view. Nor have I excluded the possible co-operation of the spinal centres. On the contrary it has been pointed out in what way they may be affected. And this way is in accord with the teachings of general pathology. Similarly it has been pointed out in what way the highest centres may have an influence, though it remains for the future to show in what way or ways their influence is actually exercised and the results of such exercise. It is to be borne in mind that if no more precise explanation is offered here neither is one offered elsewhere.

So much for general considerations in favour of the theory which I have presented. Let us now look at some particular ones.

How does this suggested theory fit in with known clinical facts? Foremost of these is the immense preponderance of attacks of acute gout in the tarso-metatarsal joint of the great toe as compared with any other joint. Any theory of gout which does not explain this fact is

inadequate, even if it is not so far as it goes erroneous. How is this explained at present. Firstly—by the remoteness of the part from the centre of circulation. Secondly—by pointing out that the whole weight of the body rests in walking upon this joint. As regards the circulation it is no more remote than that of the corresponding joints of the other toes As regards the weight of the body. In the first place, the weight of the foot, which is insignificant in proportion to the total body weight is the only burden which the toe bears in excess of that which is borne by the ankle.

But the whole weight of the body does not rest upon the great toe. It is divided between that and the other toes, the combined area of whose tarso-metatarsal joints cannot be much less, even if it is not more than that of the great toe-joint. But between this and the other toes there is this difference. The nerves around the great toe joint are not deeply seated in the soft parts, they are subject to pressure on the sole aspect by the weight of the body, on

the inner and upper aspects by the upper leather of the shoes. Whereas the nerves of the other toes which are rarely attacked are protected by the soft parts and by the cushion of the sole and are in most boots much less pressed upon than the great toe by the upper leather. I submit then that a local nerve theory of gout affords a satisfactory explanation of this clinical fact, which current views do not.

It is known that repeated attacks may occupy a great toe-joint which on subsequent examination does not show any uratic infiltration. It seems to me probable that in an ordinary attack there are two elements. An inflammation of the joint, and an inflammation of the neighbouring soft parts. On the nerve theory and having regard to the nerve distribution we can understand this and we can suspect that one element may occur without the other. It is indeed a matter of inference, rather than of observation, in many cases, that the joint has been affected at all. On the other hand we know that in old cases where the joint

destruction and infiltration proceed most actively the inflammation of the soft parts is commonly at a minimum. The same is true of the other joints. We see also uratic deposits formed in parts which have never, so far as we can judge, been the seat of any inflammatory process. Or if of any, it has been of the slightest and most transient character.

It is known that tophi are very common in the ear. This may be due partly to cold but more I think to pressure. They are usually accompanied or preceded by very slight inflammatory symptoms, often by none. The pressure is made by the weight of the head compressing them against the pillow in bed. And more especially when the ear is doubled up as often happens. We then get mechanical stress on the outside, and strain on the inside margin of the auricle. In other words pressure on one side pressure and stretching on the other.

The great infrequency of gout in the hip-joint has been commented on by writers. I suggest that this is due to the great protection from cold

and external injury afforded to its nerves by the depth at which they lie in a mass of soft parts. The sciatic nerve itself from which one at least of these nerves springs at a high level, is on the contrary much exposed to both cold and pressure and is a common seat of gouty affection. The shooting pains, the localised tendernesses are equally explicable. So also the cramps, often a most troublesome symptom.

It is well-known that the most acute attacks of toe-gout may be made to disappear with great rapidity. One method which has been adopted—amongst others by the great Harvey in his own case—is to plunge the foot into cold water. Another is by giving freely of colchicum. There are others, but these two will suffice. Now if the inflammation of the great toe be caused by a deposition of a uric salt in the cartilage, a condition which is known to be extremely stable, we have great difficulty in seeing how its removal may be effected in so short a time. On the other hand we know that the phenomena of neuritis and still more of

neuralgia are exceedingly inconstant, that they may appear and disappear spontaneously with great suddenness. We thus have an explanation, which at least is not incomprehensible, of the effect of such agents as cold and colchicum upon acute gout. To those who hold that colchicum is a drug which acts, not as an eliminant, but directly upon some part of the nervous system, this explanation appeals with superior force. It affords indeed a strong argument in support of their view. It is also worthy of notice that Weir-Mitchell insists strongly on the free application of ice in congestion of nerves.

Let us now briefly consider the bearing of this view upon the various gouty affections of internal organs. In the first place it cannot be denied that, while the existence of such disorders is fully recognised by many of us, it is very grudgingly admitted by a considerable school whose opinion is not to be treated with, nor their opposition removed by, contempt.

Now it seems to me that this opposition has in part arisen from a conception of gout,

postulating for its existence the tissues of a joint. It is from this conception that have arisen the terms of regular and irregular gout. There is not much analogy between the special tissues of a joint and those of a mucous membrane. Into a mind dominated by the idea that without a joint localised gout is not possible it is easy to see that the idea of gout in a mucous membrane will enter with difficulty. But with the admission of the idea that the starting point of a localisation of gout is an affection of the local nerves, this difficulty is removed. It enables us also to answer definitely questions which I have often had put to me by a colleague in a consultation in some such form as this. "Here is a case of bronchial catarrh, with all the ordinary physical signs and symptoms. What do you mean by saying it is 'gouty'? Do you think that all bronchial or other catarrhs are gouty?" These or suchlike questions put in good faith require an answer, which I have hitherto given as follows. "I do not consider all bronchial or other catarrhs to

be gouty; not necessarily so even when one occurs in a person who has had gout. By gouty catarrh I mean a catarrh which has for one of its factors the gouty poison in the blood. The reason I think this particular case is in this category is, firstly, because your treatment, very proper for a case of simple bronchitis, has failed. It has failed because it has not removed the gouty factor. Secondly, because there are now present, or quite recently have been, symptoms which are common in gout, from which I infer that this patient is in a gouty condition. Thirdly, I have known many cases of catarrh which have not been relieved till the treatment has been based upon this view." I should now make this explanation more definite, and therefore more acceptable by saying that the persistence of the disorder was due to the deterioration of the local nerves by the agency of the gouty poison.

This explanation is applicable to any one of the multifarious manifestations of gout. At all events in any part the nerves of which are

covered by a neurilemma. Whether or no gout poison can act directly upon naked nerves is a matter for future examination.

It is much to be hoped that the evidence of local nerve disorder in localised gout which I have collected may lead to an investigation of the state of these nerves by some of our younger pathologists, who have the necessary accomplishments and time at their disposal. Is this disorder due to neuritis, to congestion, to primary degeneration or to the changes, whatever they may be, which produce the state known to us as neuralgia. Clinical features may be recognised which might be explained by any one of these, and are especially in accord with congestion, neuralgia and neuritis.

The theory I have advocated forms a definite basis for the neural element of neuro-humoral theory of gout and brings into one line a greater number of the clinical features of gout than any other one. I commend both it and the facts on which it is based to the candid consideration of the profession.

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